Bilateral temporal lobe epilepsy

- Clinical daily rhythms of seizure in different subtypes of temporal lobe epilepsy
- Distribution of NECAB1-Positive Neurons in Normal and Epileptic Brain-Expression Changes in Temporal Lobe Epilepsy and Modulation by Levetiracetam and Brivaracetam
- Clinical characteristics of headache related to epilepsy: experience from a tertiary epilepsy center
- Semiological differences between children and adults with temporal lobe epilepsy: a video-EEG based multivariate analysis
- Lateralizing value of ictal head turning: A systematic review and meta-analysis
- Morphometric and radiomics analysis toward the prediction of epilepsy associated with supratentorial low-grade glioma in children
- Changes in topological properties of brain structural covariance networks and alertness in temporal lobe epilepsy with and without focal to bilateral tonic-clonic seizures
- Symptomatic Emotional Responses and Changes in Networks Elicited by Direct Electrical Stimulation

Bilateral Temporal Lobe Epilepsy (BTLE) is a form of drug-resistant epilepsy involving seizure activity in both temporal lobes. It is less common than unilateral temporal lobe epilepsy (TLE) and presents unique diagnostic and therapeutic challenges.

1. Clinical Features Seizure Types: Focal impaired awareness seizures (complex partial seizures) – most common, often with automatisms (lip smacking, fidgeting). Focal aware seizures (auras) – may include déjà vu, fear, auditory or visual distortions. Bilateral tonic-clonic seizures – may occur due to secondary generalization. Cognitive and Behavioral Symptoms: Memory impairment (particularly episodic memory) Emotional dysregulation (anxiety, depression) Language disturbances if involvement is asymmetric (dominant hemisphere more affected) Autonomic Symptoms: Sweating, palpitations, nausea, epigastric sensations. 2. Etiology and Pathophysiology Structural Lesions: Mesial Temporal Sclerosis (MTS) (hippocampal atrophy, gliosis, neuron loss) Bilateral cortical malformations Tumors, vascular malformations, infections Genetic Factors: Some familial cases linked to LGI1, DEPDC5, SCN1A mutations. Autoimmune Causes: Anti-LGI1, Anti-GAD, or Anti-NMDA receptor encephalitis. Post-Traumatic or Infectious (Meningitis, Encephalitis) 3. Diagnosis Video EEG Monitoring: Bilateral independent seizure onset in temporal lobes. MRI Brain: Bilateral hippocampal atrophy or other structural abnormalities. PET/SPECT: Hypometabolism or altered perfusion in both temporal lobes. Neuropsychological Testing: Confirms cognitive and memory impairments.

Treatment

A. Medical Therapy

Antiseizure Medications (ASMs): Levetiracetam, Lacosamide, Zonisamide, Clobazam Caution: Phenytoin and Carbamazepine may exacerbate bilateral onset seizures. Immunotherapy (if autoimmune suspected): IVIG, steroids, plasmapheresis.

B. Surgical Considerations

Resective Surgery: Poor candidate due to bilateral seizure foci.

see Temporal lobectomy for bilateral temporal lobe epilepsy

Neurostimulation Therapies:

Responsive Neurostimulation (RNS)

Vagus Nerve Stimulation (VNS)

Deep Brain Stimulation (DBS) – Anterior Nucleus of the Thalamus

Laser Interstitial Thermal Therapy (LITT): Experimental in bilateral cases.

Prognosis

Poorer seizure control compared to unilateral TLE. High risk of cognitive decline if seizures remain uncontrolled. Best outcomes with early diagnosis and multimodal treatment.